Supporting Information

Wayman *et al.* 10.1073/pnas.0803072105

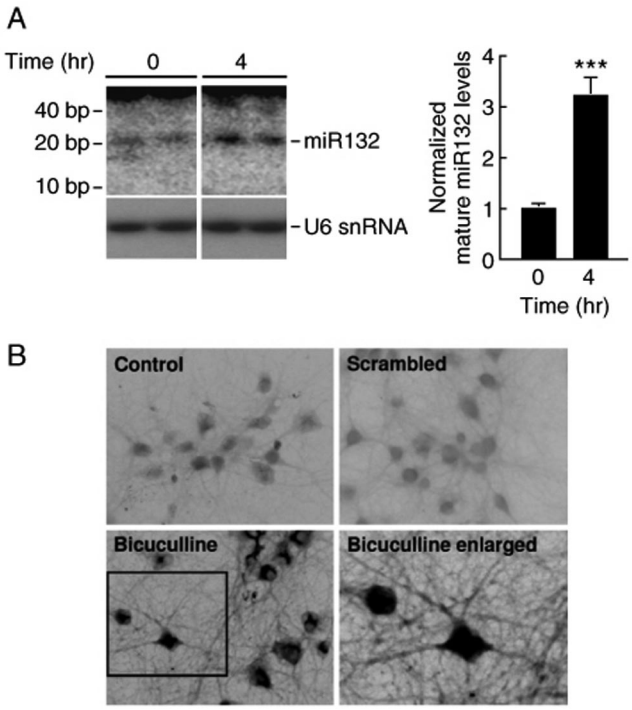

Fig. S1. (*A*) Bicuculline increases miR132 levels. Hippocampal neurons were stimulated with 20 μM bicuculline for 4 h. After incubation, the neurons were lysed, and total RNA was isolated. Densitometric quantitation of miR132 levels from six biological replicates are shown on the right. (*B*) *In situ* analysis of hippocampal neurons stimulated with 20 μM bicuculline. Staining in both soma and dendrites was detected. Control represents unstimulated neurons ($\pm$SEM, ***, $P < 0.001$).

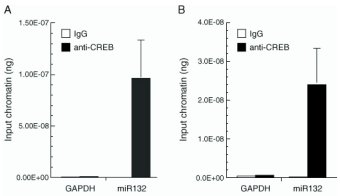

Fig. S2. Chromatin from hippocampal neurons (*A*) or hippocampi of 12-week-old mice (*B*) was subjected to ChIP using a CREB antibody or IgG control. Real-time PCR was conducted using primers that interrogate the miR132 gene promoter.

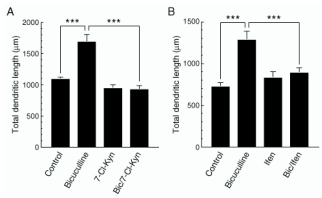

Fig. S3. Activity-dependent dendritic growth requires functional NMDA receptors. Hippocampal neurons expressing MAP2B-EGFP were treated with ±20 μM bicuculline for 48 h ± 100 μM 7-Cl-kynurenic acid (*A*) and ± 3 μM ifenprodil (*B*). Dendritic length was quantified at 9 DIV (±SEM, ANOVA; ***, $P < 0.001$).

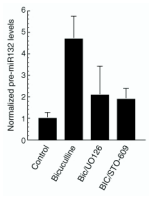

Fig. S4. Activity-dependent miR132 induction requires the CaM kinase and MAP kinase cascades. Hippocampal slices from postnatal day 5 rats were cultured for 4 days, pretreated with either 5 μM STO-609 or 10 μM UO126 for 4 h, and stimulated with $\pm$20 μM bicuculline for 4 h. RNA was reverse-transcribed and analyzed by real-time PCR with premiR132 cDNA primers. The data were normalized to GAPDH cDNA levels ($\pm$SEM, $n = 5–6$).